Original Article

Correlation of end-tidal CO2 (ETCO2) level with serum lactate in patient with septic shock

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Abstract

Background: *Septic shock patients usually have perfusion disturbance that causes increased lactate production, induces metabolic acidosis. This is compensated by hyperventilation. As the consequence decreased $PaCO_2$ can be measured as end-tidal CO_2 ($ETCO_2$). Thus monitoring of End Tidal carbon dioxide ($ETCO_2$) could be a convenient monitoring in Septic Shock*

Objectives: *To see the correlation between end-tidal CO_2 with serum lactate in Septic Shock patient.*

Methods: *This was observational, cross sectional study conducted on January to July, 2020 at ICU in Shaheed Suhrawardy Medical College Hospital. Subjects were septic shock patient aged between two extreme age recruited by purposive sampling.$ETCO_2$ measured by capnograph, Serum lactate and blood gas analysis were done. Clinical and biochemical data were recorded and analyzed. P value was determined by Pearson correlation. P value was significant at <0.05.*

Results: *There were 80 subjects analyzed with median age 40 years old. Most of the patients had compensated metabolic acidosis. Correlation analysis between $ETCO_2$ and lactate showed significant strong negative correlation (r= -0.747; p=0.001).Using Pearson correlation, an inverse relationship was noticed between serum lactate and $ETCO_2$ (p<0.0001, r=-0.65).*

Conclusion: *In septic shock patient increase serum lactate level was correlated with decrease $ETCO_2$ on admission and $ETCO_2$ measurement by capnography is a non-invasive procedure and it takes less time.*

Key words: *End-tidal Carbon dioxide, Hyperlactatemia, Septic shock*

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Introduction

Septic shock is responsible for significant mortality among patients admitted to the ICU[1,8]. Despite many advances in our understanding and treatment, epidemiologic studies across the globe have shown an increasing incidence of septic shock patient with continued high mortality rate. Early identification and treatment of septic shock have shown to improve survival[2]. So to improve the standard care for septic shock some guidelines are developed to predict severity of septic shock[3,4]. A hall mark of septic shock is hypoperfusion leading to end organ damage[1,3]. This eventually led to imbalance of production and elimination of lactate which causes hyperlactatemia[9]. So serum lactate level becomes a useful marker for resuscitation of septic shock and shown to predict mortality in critical patient [5,6]. Using serum lactate as a diagnostic procedure takes time approximate 172 minutes in emergency setting[7]. This could result in treatment delay for septic shock. Mortality rate have shown to decrease as much as 16 percent when early goal directed therapy is initiated[2]. Although Lactate level provides a reliable insight into disease severity they may not be rapidly or easily attainable. In septic shock patient compensation to lactic acidosis hyperventilation occurs and causes decrease CO_2 alveolar pressure. Based on this association $ETCO_2$ measurement can be used as an early identification of septic shock[11]. Capnography, a noninvasive, real time method of determining $ETCO_2$ has been shown to predict severity of septic shock[8,9,11]. We examine patient with septic shock to investigate the association of $ETCO_2$ with mortality and serum lactate. The aim of this study is to search the correlation between $ETCO_2$ and serum lactate in patients with septic shock.

Materials & Methods

The study was conducted in the Department of Anaesthesiology in Shaheed Suhrawardy Medical College Hospital (ShSMCH), Dhaka from 1st January, 2020 to 31st July, 2020. Study population was patients with septic shock aged in between two extreme ages with mean arterial pressure less than 65 mmHg with or without ionotrope. This was observational, cross sectional study. Estimated sample size is 80. Purposive sampling was done. Exclusion criteria were not willing to participate in this study, patients with preexisting pulmonary disease (Asthma or COPD), hyperthermia from environmental cause, Two extreme age group, Intubated patient. Main outcome variables were serum lactate, $ETCO_2$ and mortality in septic shock patients. Laboratory test were done serum lactate and arterial blood gas analysis. Written informed consent was taken. We collect base line inclusion criteria data with detailed medical history and printed on collection form. After data collection, data editing and clearing was done manually and prepared for data entry and analysis by using SPSS (statistical package for social science).

Results

In this study enrolled patients mean age was 42.2 years and male patients are more. Septic shock patients showed higher heart rate and respiratory rate, lower systolic pressure and diastolic pressure. Regarding co morbidities 23.8% patients had DM, 16.3% had CKD, 11.3% had HTN and 7.5% had other. Mortality among the patients with history of co morbidities had no significant difference. Higher mean serum lactate and lower mean $ETCO_2$ on admission was seen in non surviving patients. The mean serum lactate was found 1.45±0.76 mmol/L with range from 0.5 to 3.6 mmol/L. The mean $ETCO_2$ was found 30.8±7.2 mmHg with range from 15.7 to 46.3 mmHg. In this study septic shock patients with $ETCO_2$ level ≤20 mmHg had their mean serum lactate 2.92±0.58 mmol/L. some patients had $ETCO_2$ level 21-25 mmHg and their mean serum lactate was 2.07±0.73 mmol/L. $ETCO_2$ level 26-30 mmHg found in septic shock patients with their mean serum lactate was 1.52±0.56 mmol/L and 31-35 mmHg was seen where mean serum lactate was 1.08±0.34 mmol/L. 51.25% patients were defined as early septic shock and 48.75% were late septic shock. 85.0% patients had metabolic acidosis and 15.0% had without metabolic acidosis. 56.25% patients were improved and 43.75% were death in-hospital. Association between outcome with co morbidities had no statistical significant ($P > 0.5$). Association with serum lactate level and $ETCO_2$ had found statistically significant ($P < 0.5$). 85.7% patients were late septic shock in death group and 20.0% in improved group. The difference was statistically significant ($p<0.05$) between two groups. Mean $ETCO_2$ was found 30.6±7.2 mmHg in metabolic acidosis group and

31.8±7.0 mmHg in without metabolic acidosis group. 85.7% patients had metabolic acidosis in death group and 84.4% in improved group. The difference was not statistically significant (p>0.05) between two groups. Here mean $ETCO_2$ was found 30.2±8.1 mmHg in hospital stay ≤10.0 days and 31.2±6.4 mmHg in hospital stay >10.0 days. 54.3% patients was found hospital stay ≤ 10.0 days in death group and 33.3% in improved group. The difference was statistically significant (p<0.05) between two groups. However, Hospital stay (≤10 days) was not found to be independent predictor for in-hospital death.

Table 1: Demography, Comorbidities & S.lactate, ETCO2 level in this Study population.

characteristics	N(80)
Age (Mean±SD)	43.8±7.6
Male	57.5%
female	42.5%
Mean arterial pressure	62.3± 2.3 mmHg
Co morbidities	
Diabetes	23.8%
Chronic kidney disease	16.3%
Hypertension	11.3%
Others	7.5%
Serum Lactate and $ETCO_2$ level of study population	
Serum lactate (Mean±SD)	1.45±0.76
$ETCO_2$ (Mean±SD)	30.8±7.2
Outcome	
Improved	56.25%
Death	43.75%

Table 2 : Serum lactate in different $ETCO_2$ level (n=80)

$ETCO_2$ (mmHg)	**n**	**Serum lactate (mmol/L)**	
		Mean±SD	**Range (min-max)**
≤20	7	2.92±0.58	2.1-3.6
21-25	12	2.07±0.73	0.9-3.6
26-30	19	1.52±0.56	0.7-2.5
31-35	20	1.08±0.34	0.7-1.8
36-40	15	0.94±0.23	0.5-1.3
>40	7	0.92±0.23	0.5-1.2

Table 3 : Association between outcome with serum lactate (n=80)

	Total (n=80)	**Outcome**		**P value**
		Death (n=35)	**Improved (n=45)**	
	Mean ±SD	**Me±SanD**	**Me±SanD**	
Serum lactate (mmol/L)	1.45±0.76	1.7±0.787	1.2±0.154	0.001[s]
Range (min-max)	0.5-3.6	0.7-3.6	0.5-2.5	

s= significant
P value reached from unpaired t-test

Table 4 : Association between outcome with end tidal CO_2 (n=80)

	Total (n=80)	**Outcome**		**P value**
		Death (n=35)	**Improved (n=45)**	
	Mean±SD	**Me±SanD**	**Me±SanD**	
$ETCO_2$ (mmHg)	30.8±7.2	28.±7.27	32.8±6.1	0.004[s]
Range (min-max)	15.7-46.3	15.44.75	19.-746.3	

s= significant
P value reached from unpaired t-test

Table 5 : Multivariate regression analysis for in-hospital death (n=80)

	Adjusted OR	**95% CI**		**P Value**
		Lower	**Upper**	
Abnormal serum lactate (mmol/L)	3.365	1.138	9.952	0.028[s]
Abnormal $ETCO_2$ (mmHg)	2.499	1.017	8.031	0.046[s]
Hospital stay (≤10 days)	2.667	0.991	7.180	0.052[ns]

Discussion

Patients with septic shock tissue perfusion decreases and it produce lactic acid due to anaerobic metabolism. A multicentre study by khosravani H et al[10] showed hyperlactatemia is frequent in critically ill patients. Interestingly my findings match with them. And as a compensation hyperventilation occurs and it causes decrease CO_2 alveolar pressure. Christopher L et al observed that low $ETCO_2$ level were the strongest predictor of sepsis. They showed $ETCO_2$ is associated with mortality and lactate in patients with suspected sepsis[12]. In septic shock patient using Pearson correlation calculation showed that increase serum lactate level was correlated with decrease $ETCO_2$ on admission

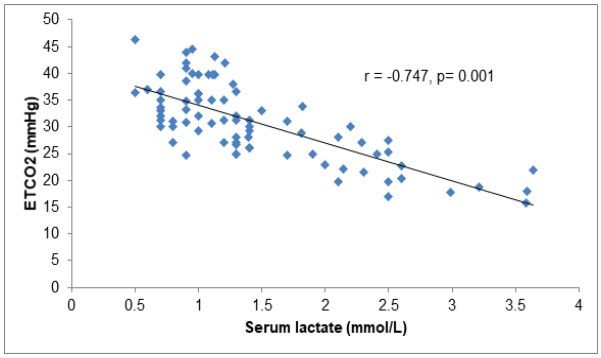

Figure: Pearson correlation showing negative correlation (r= -0.747; p=0.001) between $ETCO_2$ and serum lactate level.

In this study observed that multivariate analysis shows abnormal serum lactate and abnormal $ETCO_2$ were found to be independent predictors for hospital mortality in septic shock. And $ETCO_2$ measurement by capnography is a non-invasive procedure. It takes less time in comparison to serum lactate. On admission early goal directed treatment is possible.

Conclusion

In conclusion, End tidal CO_2 ($ETCO_2$) level was related to serum lactate in patient with septic shock. $ETCO_2$ measured by capnograph is a non-invasive and fast method to detect hyperlactatemia where lactate level does not available.

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